

Patent claims

1. The use of at least one substance for detecting the expression and/or function of activated and/or inactive Sgk, in particular Sgk1 and/or Sgk3, and/or PKB, and/or Nedd, in particular Nedd4-2, for the purpose of diagnosing diseases which are associated with disturbed glucose transport.
2. The use as claimed in claim 1, characterized in that the substance is at least one substance from the group of antibodies and nucleotides.
3. The use as claimed in claim 1 or claim 2, characterized in that use is made of antibodies which are directed against phosphorylated and/or unphosphorylated sequences in Sgk, in particular Sgk1 and/or Sgk3, PKB and/or Nedd, in particular Nedd4-2.
4. The use as claimed in claim 3, characterized in that use is made of antibodies which are directed against at least one phosphorylated and/or unphosphorylated kinase consensus sequence, in particular an Sgk1 consensus sequence, in a Nedd protein, in particular in the Nedd4-2 protein.
5. The use as claimed in one of the preceding claims, characterized in that at least one mutation, in particular an inactivating mutation, is detected in Nedd, in particular in nedd4-2, in DNA, RNA and/or a Nedd protein from a biological sample, in particular a sample from a patient, with the mutation preferably being present in a segment of nedd which encodes an Sgk1 consensus sequence in the Nedd protein.

6. The use as claimed in claim 5, characterized in that the mutation is ^{S338D}Nedd4-2 and/or ^{S444D}Nedd4-2.
7. The use as claimed in one of the preceding claims, characterized in that at least one mutation, in particular an activating mutation, is detected in *sgk*, in particular in *sgk1* and/or *sgk3*, and/or a gene for PKB, in DNA, RNA and/or an Sgk protein and/or PKB protein from a biological sample, in particular a sample from a patient.
8. The use as claimed in claim 7, characterized in that the mutation is ^{S422D}Sgk1 and/or ^{T308D, S473D}PKB.
9. The use as claimed in one of the preceding claims, characterized in that the diseases are the metabolic syndrome, in particular obesity.
10. A method for diagnosing predispositions to obesity, characterized in that at least one polymorphism is detected in *sgk*, in particular *sgk1* and/or *sgk3*, a gene for PKB, *nedd*, in particular *nedd4-2*, and/or *sglt*, in particular *sglt1*.
11. The method as claimed in claim 10, characterized in that the polymorphism is a single nucleotide polymorphism (SNP).
12. The method as claimed in claim 10 or claim 11, characterized in that the polymorphism is E8CC/CT;I6CC in *sgk1*.
13. The use of at least one active compound for exerting an effect on glucose transport, in particular intestinal and/or renal glucose transport.

14. The use as claimed in claim 13, characterized in that the active compound exerts an effect on at least one Sgk, in particular Sgk1 and/or Sgk3, and/or PKB, and/or an effect on at least one Nedd, in particular Nedd4-2.
15. The use as claimed in claim 13 or claim 14, characterized in that the active compound is directed against an Sgk, in particular Sgk1 and/or Sgk3, and/or PKB and/or a Nedd, in particular Nedd4-2.
16. The use as claimed in one of claims 13 to 15, characterized in that the active compound is directed against activators, inhibitors, regulators and/or biological precursors of an Sgk, in particular of Sgk1 and/or Sgk3, and/or PKB and/or a Nedd, in particular Nedd4-2.
17. The use as claimed in one of claims 13 to 16, characterized in that the active compound is a polynucleotide which preferably encodes a peptide, in particular a polypeptide.
18. The use as claimed in one of claims 13 to 17, characterized in that the active compound is a peptide, preferably a polypeptide.
19. The use as claimed in claim 17 or claim 18, characterized in that the peptide exerts an effect on the expression and/or function of an Sgk, in particular Sgk1 and/or Sgk3, and/or PKB and/or a Nedd, in particular Nedd4-2.
20. The use as claimed in one of claims 13 to 19, characterized in that the active compound is a "small molecular compound", preferably a "small

molecular compound" having a molecular weight (MW) of < 1000.

- 5 21. The use as claimed in one of claims 13 to 20, characterized in that the active compound inhibits at least one Sgk, in particular Sgk1 and/or Sgk3, and/or PKB, and/or stimulates at least one Nedd, in particular Nedd4-2, in particular for the purpose of preventing or treating diseases which are connected with disturbed glucose absorption.
- 10 22. The use as claimed in one of claims 13 to 21, characterized in that the active compound is at least one kinase inhibitor, preferably staurosporine and/or chelerythrine, or one of their analogs, and/or at least one ligase activator.
- 15 23. The use of at least one active compound for exerting an effect on, in particular inhibiting, at least one Sgk and/or PKB, and/or for exerting an effect on, in particular stimulating, at least one Nedd, for the purpose of producing a drug or a pharmaceutical composition for treating diseases which are connected with disturbed glucose transport.
- 20 24. The use as claimed in one of claims 21 to 23, characterized in that the diseases are the metabolic syndrome, in particular obesity.
- 25 25. The use as claimed in one of claims 13 to 20, characterized in that the active compound stimulates at least one Sgk, in particular Sgk1 and/or Sgk3, and/or PKB, and/or inhibits at least one Nedd, in particular Nedd4-2, for the purpose of increasing glucose transport, in particular for increasing the bodyweight of animals.
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26. The use as claimed in claim 25, characterized in that the active compound is at least one Sgk activator and/or PKB activator, in particular a growth factor, preferably IGF1, and/or insulin.
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27. The use as claimed in claim 25 or claim 26, characterized in that the active compound is at least one stimulant of the transcription of sgk1 and/or sgk3 and/or a gene for PKB, preferably at least one glucocorticoid, mineral corticoid, gonadotropin and/or cytokine, in particular TGF β .
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28. A diagnostic kit which comprises at least one substance for detecting the expression and/or function of activated and/or inactive Sgk, in particular Sgk1 and/or Sgk3, and/or PKB and/or Nedd, in particular Nedd4-2, for diagnosing diseases which are associated with disturbed glucose transport.
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29. The diagnostic kit as claimed in claim 28, characterized in that the diseases are the metabolic syndrome, in particular obesity.
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30. An antibody, characterized in that it is directed against at least one phosphorylated kinase consensus sequence, in particular an Sgk1 consensus sequence, in a Nedd protein, in particular in the Nedd4-2 protein.
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31. An antibody, characterized in that it is directed against at least one unphosphorylated kinase consensus sequence, in particular an Sgk1 consensus sequence, in a Nedd protein, in particular in the Nedd4-2 protein.
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32. An antibody, characterized in that it is directed against at least one mutated kinase consensus
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sequence, in particular an Sgk1 consensus sequence, in a Nedd protein, in particular in the Nedd4-2 protein.

- 5 33. The antibody as claimed in claim 32, characterized in that the Nedd protein with a mutated kinase consensus sequence is ^{S338D}Nedd4-2 and/or ^{S444D}Nedd4-2.
- 10 34. A composition, in particular a pharmaceutical composition, comprising an effective quantity of at least one active compound which exerts an effect on glucose transport, in particular intestinal and/or renal glucose transport, and, 15 where appropriate, a pharmaceutically acceptable excipient.
- 20 35. The composition as claimed in claim 34, characterized in that the active compound exerts an effect on at least one Sgk and/or PKB and/or at least one Nedd.
- 25 36. The composition as claimed in claim 34 or claim 35, characterized in that the active compound exerts an effect on activators, inhibitors, regulators and/or biological precursors of an Sgk, in particular of Sgk1 and/or Sgk3, and/or PKB and/or a Nedd, in particular Nedd4-2.
- 30 37. The composition as claimed in one of claims 34 to 36, characterized in that the active compound is a polynucleotide which preferably encodes a peptide, in particular a polypeptide.
- 35 38. The composition as claimed in one of claims 34 to 37, characterized in that the active compound is a peptide, preferably a polypeptide.

39. The composition as claimed in claim 37 or claim 38, characterized in that the peptide exerts an effect on the expression and/or function of an Sgk, in particular Sgk1 and/or Sgk3, and/or PKB and/or a Nedd, in particular Nedd4-2.
40. The composition as claimed in one of claims 34 to 39, characterized in that the active compound is a "small molecular compound", preferably a "small molecular compound" having a molecular weight (MW) of < 1000.
41. The composition as claimed in one of claims 34 to 40, characterized in that the active compound inhibits at least one Sgk and/or PKB and/or stimulates at least one Nedd.
42. The composition as claimed in one of claims 34 to 41, characterized in that the active compound is at least one kinase inhibitor, preferably staurosporine and/or chelerythrine or one of their analogs, and/or at least one ligase activator.
43. The composition as claimed in one of claims 34 to 42, characterized in that the active compound stimulates at least one Sgk and/or PKB and/or inhibits at least one Nedd.
44. The composition as claimed in claim 43, characterized in that the active compound is at least one Sgk activator and/or PKB activator, in particular a growth factor, preferably IGF1, and/or insulin.
45. The composition as claimed in claim 43 or claim 44, characterized in that the active compound is at least one stimulant of the transcription of sgk1 and/or sgk3 and/or a gene for PKB, preferably at

least one glucocorticoid, mineral corticoid, gonadotropin and/or cytokine, in particular TGF β .

- 5 46. A method for producing transgenic animals, excluding humans, which exhibit an increase in lipid deposition in adipose tissue, characterized in that the expression and/or function of Sglt, in particular Sglt1, is increased.
- 10 47. The method as claimed in claim 46, characterized in that Sglt, in particular Sglt1, is over-expressed.
- 15 48. The method as claimed in claim 46 or claim 47, characterized in that the expression and/or function of at least one Sgk, in particular Sgk1 and/or Sgk3, and/or PKB, is increased.
- 20 49. The method as claimed in claim 48, characterized in that at least one sgk, in particular sgk1 and/or sgk3, and/or at least one gene for PKB, is overexpressed.
- 25 50. The method as claimed in claim 48 or claim 49, characterized in that use is made of at least one activating mutation of sgk, in particular of sgk1 and/or sgk3, and/or of a gene for PKB, in particular ^{S422D}sgk1 and/or ^{T308D, S473D}PKB.
- 30 51. The method as claimed in one of claims 46 to 50, characterized in that the expression and/or function of at least one Nedd, in particular Nedd4-2, is decreased.
- 35 52. The method as claimed in claim 51, characterized in that use is made of at least one inactivating mutation of nedd, in particular of nedd4-2, in particular ^{S338D}nedd4-2 and/or ^{S444D}nedd4-2.